geal junction. The slit was repaired in layers with fine cotton sutures. The mediastinum was left open and the chest was closed. Two tubes, one anterior and one posterolateral, were placed for drainage. A Levine tube had previously been placed under direct vision into the stomach for decompression.

Gastric suction and water seal drainage were provided and penicillin, streptomycin and aureomycin were given parenterally along with supportive fluids for the following several days. The patient recovered gradually and was discharged on the 18th postoperative day. She was taking an adequate soft diet by the seventh postoperative day.

DIAGNOSIS

The symptoms of perforation of the esophagus are so severe that it is obvious both to the patient and the physician that a catastrophic incident has occurred. Although the symptoms usually are such as to indicate an upper abdominal lesion, the associated dyspnea and cyanosis suggest difficulty above the diaphragm. The pain is sudden and excruciating, usually follows a sudden effort of regurgitation and may be described as epigastric or substernal. Pain in the lower thoracic region of the back is common, as is pain low in the left side of the chest. Mediastinal emphysema (as was noted in Case 2) and palpable crepitation in the neck later in the course of the disease may be noted. Roentgenograms may reveal evidence of fluid or air in the pleural cavity. This was observed in both of the cases here reported. Swallowed Lipiodol® may extravasate into the mediastinum or pleural cavity, further confirming the diagnosis. Aspiration of gastric contents from the pleural cavity may give confirmatory evidence if the mediastinal pleura has been ruptured, as it was in both of the cases reported here.

TREATMENT

All recent communications[3, 4, 6, 7] have emphasized the need for immediate surgical repair. The critical appearance and state of the patient should present no contraindication to thoracotomy after the institution of rapid measures toward supportive treatment. These initial measures consist of oxygen inhalation, infusion of plasma or whole blood, administration of antibiotics and, in the presence of tension or excessive amounts of fluid, aspiration of the pleural cavity.

Recovery following late drainage of the pleural cavity has been reported in a few cases. This procedure should be reserved for misdiagnosed cases in which the patient is seen late in the course of the illness following an initial spontaneous improvement.

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Cat Scratch Disease on the San Francisco Peninsula

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CAT SCRATCH DISEASE, or benign lymphoreticulosis, is a disorder first recognized by Foshay of Cincinnati in 1932. He observed certain cases of lymphadenitis in which the pus was always sterile and in which serological studies were negative for tularemia. A history of cat bite or scratch, or of close association with cats, was obtained in all these cases. The observations were never published.

In 1945 Hangar of New York studied an apparent occurrence of this disease in himself. Paronychia developed and regional adenitis, malaise and fever ensued. Rose and Hangar prepared an antigen which, when it was injected intradermally, evoked intense reaction similar to the response to tuberculin. With the same antigen they obtained positive reaction in patients formerly studied by Foshay. All attempts to inoculate hen's eggs and transmit the disease to animals failed. In Hangar's case there was no known bite or scratch from the family cat, but there was intimate contact with the animal at the time paronychia developed.

When in 1947 Debré of Paris visited Foshay, he learned of the cat scratch disease entity and of the intradermal test with the specific antigen. Having seen similar cases in Paris, he took some antigen with him on his return and was able to prove the identity of the disease with that described by Foshay. His report, La Maladie des Griffes de Chat,[4] was the first that was published. Since then many cases have been reported from Paris. The first report in English was made by Greer and Keefer in 1951.[7] Daniels and MacMurray of the District of Columbia made extensive studies and published two papers.[2,3] There

From the Stanford University Health Service and The Palo Alto Clinic.

have been three reports of cases in California, by Gifford and co-workers,[6] by Epstein[5] and by Cuttle,[1] all published in 1952. So far no cases have been reported from the San Francisco Peninsula.

It should be noted that although the cat plays an important part in dissemination, the animal itself has never been found to show any signs of disease, and the skin test with specific antigen gives no reaction.

The disease has not yet been etiologically defined but it is presumed that the causative factor is a large virus related to the psittacosis lymphogranuloma group. There is usually a tendency to slight cross-agglutination with psittacosis antigen. The disease has occurred in patients from 18 months to 57 years of age.

REPORT OF A CASE

The patient, a man 29 years of age, reported to the Health Service, Stanford University, on May 5, 1952, because of a lump just above the right elbow. He felt well and there were no symptoms of acute infection. No enlargement of axillary nodes was noted. There was a small scratch with indolent infection on the dorsum of the third finger of the right hand. No treatment was given but the patient was told to return if the lump did not recede. Ten days later he returned because the mass above the elbow was considerably larger and on this visit two small nodes were felt in the right axilla. The indolent lesion on the finger was unchanged. Although the patient had had no malaise and no fever that he was aware of, when inquiry elicited that there were two house cats to which he was exposed, the possibility of cat scratch disease was considered. The hemoglobin content of the blood was 15 gm. per 100 cc. Erythrocytes numbered 5 million per cu. mm. and leukocytes 4,900—49 per cent lymphocytes, 47 per cent neutrophils, 2 per cent basophils, 1 per cent monocytes and 1 per cent eosinophils. The sedimentation rate was 6 mm. in one hour. Heterophil antigen agglutination and the result of a Wassermann test were negative.

On June 6 an intradermal test with cat scratch disease antigen was carried out and 48 hours later there was a 4 mm. papule with a 2.5 cm. pink areola at the site of injection, which was interpreted as a strongly positive reaction. The epitrochlear mass by then was greatly enlarged and fluctuant. At the center the skin was dark pink, and from there it faded gradually to a violaceous color at the periphery. The epidermis over the area of deepest color was scaling (see Fig. 1). At this time 40 cc. of sanguinopurulent exudate was withdrawn. The axillary nodes had coalesced and the mass was approximately 4 cm. in diameter, but was not fluctuant.

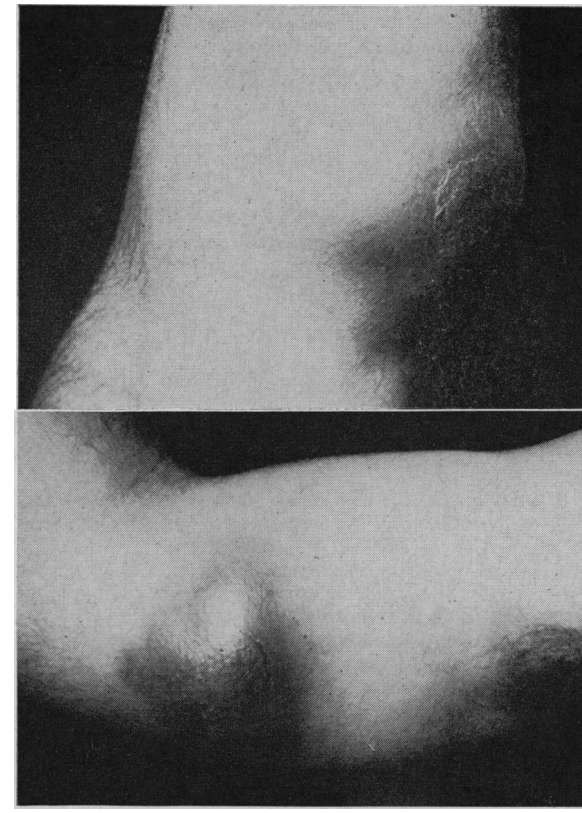

Figure 1.—*Upper*—Mass above the elbow at time of greatest enlargement and discoloration, June 6. *Lower*—On June 12 the mass was somewhat smaller than on June 6. This picture was made six days after first aspiration and just before the second.

No evidence of disease of the hilar nodes was seen in a roentgenogram of the chest taken June 9. On June 12 tuberculin, histoplasmin and coccidioidin skin tests were done and the results were negative. On June 16 agglutination for tularemia was negative. Complement fixation for Q fever was negative in all dilutions. Complement fixation with psittacosis antigen* was strongly positive in dilution 1:2 through dilution 1:16, equivocal at 1:32 and negative in greater dilution. Two weeks later there was a strongly positive reaction extending through dilution 1:32, a 1 plus reaction at 1:64 and negative result in higher dilutions.

Material was aspirated from the epitrochlear mass four times in 18 days and 94 cc. was withdrawn. The axillary tumor was aspirated twice and 8 cc. of yellowish purulent material was removed. The pus was sterile aerobically and anaerobically. Results of inoculation of guinea pigs, white rats and macacus rhesus monkeys were negative. An antigen was prepared from the material† and later used for intra-

*Through the courtesy of the Hooper Foundation.

†Courtesy of Dr. Houghton Gifford, Stanford Medical School.

dermal test on the patient, and again there was a very strongly positive reaction.

The masses gradually diminished and at last disappeared.

SUMMARY

A case of cat scratch disease in a resident of the San Francisco Peninsula, with suppuration of regional lymph nodes, is reported.

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Cat Scratch Disease in the Los Angeles Area

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SINCE THE ORIGINAL REPORTS of cat scratch disease in Europe in 1950[2] and this in country in 1951[5] cases have been reported from many places, particularly from eastern, middle western and southern areas of the United States. Several cases in California have been reported[1, 3] and others diagnosed but not reported.[6, 8]

Several other descriptive terms have been used for the disease, such as cat scratch fever, benign inoculation lymphoreticulosis, cat scratch syndrome, benign lymphoreticulosis of inoculation, and possibly others. However, the uniformity of symptoms and data in reported cases seems to warrant the name *cat scratch disease.*

It is a benign self-limited disease, apparently of viral origin.[7] Characteristic features are tenderness of regional lyph nodes in association with systemic symptoms such as fever and malaise (rarely, cutaneous manifestations) that develop some two to three weeks after the appearance of a local, encrusted, oozing lesion at the site of a scratch or a bite by a cat or a break in the skin of a person who handles cats.

Much research on laboratory procedures for aid in diagnosis has shown that results of various kinds of determinations are essentially within normal limits, although in many cases the number of eosinophils in the blood is high in relation to the total number of leukocytes. The diagnosis can readily be established, however, by observing the reaction to intradermal injection of an antigen prepared from a suppurative node, or by biopsy of such a node.[4]

REPORT OF A CASE

A girl five and a half years of age was first observed in the office of one of the authors in Sun Valley, California (near Los Angeles) on October 13, 1952, with complaint of fever, slight nuchal rigidity and tenderness of axillary lymph nodes. Upon physical examination a small fresh scar on the left side of the chest was noted, and the lymph nodes in the left axillary region were enlarged, very tender and appeared to be matted together rather than separate and discrete. The temperature was 100° F. Penicillin, 450,000 units, was injected intramuscularly and a triple sulfonamide, gm. 0.25 by mouth four times daily. Three days later the axillary mass was larger, definitely matted and quite tender. No abnormalities were noted on examination of the blood or on urinalysis. Aureomycin, 100 mg. three times daily by mouth, was prescribed. When the patient was next examined four days later the nodes, still tender and undiminished in size, felt fixed in place. Aureomycin was continued. On October 27 the nodes were of the same size but the tenderness had subsided and surgical biopsy was advised.

The patient entered the hospital October 30. The hemoglobin content of the blood was 12.1 gm. per 100 cc. and erythrocytes numbered 4.3 million per cu. mm. Leukocytes numbered 7,000 per cu. mm.—41 per cent neutrophils, 48 per cent lymphocytes, 4 per cent monocytes and 7 per cent eosinophils. Results of urinalysis were normal.

A 4x3x1.5 cm. mass of tissue including four lymph nodes was removed for pathologic study.

PATHOLOGIST'S REPORT

The specimen was a mass of fibrofatty tissue with several lymph nodes imbedded in it, the largest of them 1 cm. in diameter. On microscopic examination of the lymph nodes it was noted that there were numerous semicaseous, necrotic foci containing a few polynuclear cells and surrounded by heavy mantles of epithelioid cells with occasional giant cells. No acid-fast organisms were observed. The diagnosis was granulomatous lymphadenitis consistent with cat scratch disease.

When questioned specifically the parents of the patient recalled that a cat had scratched her on the